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THE DYNAMIC ELASTIC PROPERTIES OF THE ARTERIAL WALL

By D. H. BERGEL

From the Department of Physiology, St Bartholomew's Hospital Medical College, London, E.C. 1

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Although the response of the arterial tree to relatively slow changes in blood pressure is determined by its static elastic properties (Bergel, 1961), the rapid pressure changes occurring at each heart beat will result in rather different behaviour. This is due to the visco-elastic properties of the arterial wall. The mechanical response of a visco-elastic material depends both on the force applied (elastic response) and on the time it acts (viscous response). These substances display 'creep' (continuing extension at constant load) and stress relaxation (tension decay at constant length). The properties of such a material can be defined by measurements of either of these phenomena, but it is difficult to infer from this the response to a rapid stress. A more direct form of enquiry is to measure the oscillatory stress–strain relationships at any required frequency, which has previously been done with strips and rings cut from arteries and recently with intact vessels *in vivo* (Petersen, Jensen & Parnell, 1960). When using intact vessels it is necessary to measure simultaneously the amplitude and the phase relationships of oscillatory pressure and radius changes. As arteries change very little in length with each heart beat (Lawton & Greene, 1956) they should be held at their natural length.

METHODS

The apparatus used is shown diagrammatically in Fig. 1; its use has been explained previously (Bergel, 1961). Changes in the radius of the specimen are detected with a collimated light beam and a photomultiplier tube (R.C.A. 931A).

Sinusoidal variations in volume were superimposed upon that due to a steady pressure of 100 mm Hg by a cam-operated pump (Taylor, 1959) leading to the vessel through the lower of the two supports. The pressure changes developed were detected by a capacitance manometer (Southern Instruments) attached to a wide cannula. This was inserted through the upper support and its tip lay in the vessel at the level where radius changes were detected by the light beam.

To determine the amplitude and phase distortion the manometer was calibrated dynamically, both by the pressure transient method of Hansen (1949) and by direct measurement. The latter was achieved by comparing its output at frequencies of 2–25 c/s with that from an air-filled unit with a natural frequency of 940 c/s. The manometer and cannula used in

these experiments was found to have an undamped natural frequency of 89 c/s and a degree of damping of 0·102. According to Hansen's formulae it should, at 20 c/s, over-register pressure changes by 5·2% with a phase lag of 0·048 radians. This was confirmed, within the limits of error, by the direct measurements. Accordingly the appropriate corrections were made to all readings.

Automatic Fourier analysis of the signals representing pressure and radius changes was performed by a resolver (Taylor, 1959), synchronized by a switch-plate mounted on the cam-shaft of the pump. Direct readings of the sine and cosine coefficients of both variables could be made at each frequency.

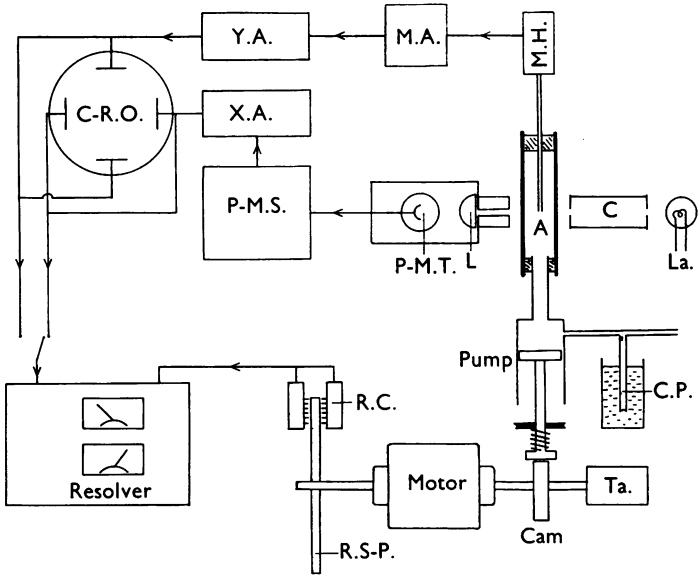

Fig. 1. Formalized diagram of the apparatus. A, arterial specimen; C, collimating tube; La, light source; C.P., mercury-column manostat; Ta, tachometer; R.S-P., resolver switch-plate; R.C., resolver contacts; L, lens; P-M.T., photomultiplier tube; P-M.S. photomultiplier power supply; M.H., manometer transducer head; M.A. manometer amplifier; X.A., X amplifier; Y.A., Y amplifier; C-R.O., cathode-ray oscilloscope.

Dynamic measurements were normally made after determining the static stress–strain relationships (Bergel, 1961) but in some cases the dynamic experiments alone were performed. These vessels were held at a steady pressure of 100 mm Hg for some minutes to allow stress relaxation.

The specimens used were dog's thoracic and abdominal aorta, femoral and carotid arteries, removed and studied within a few hours of death by bleeding. These were filled with isotonic saline at room temperature. The frequency range covered was from 2 to 18 c/s.

The markedly non-linear properties of the arterial wall (Bergel, 1961) lead to two difficulties. In the first place, a fall in mean pressure due to a small leak will result in a diminished elastic modulus. As small a change as 2 mm Hg could be detected in this way. To overcome this the barrel of the pump was connected, by a narrow tube, to a saline reservoir held at a constant pressure of 100 mm Hg by a mercury column manostat (Fig. 1, C.P.). This also altered the magnitude and phase of the pressure changes, but since the radius changes were similarly affected (as was shown by direct experiment) no errors resulted.

Secondly, the non-linearity of the wall will distort the pressure changes generated by a sinusoidal alteration in volume. However, provided the oscillations are kept small enough, this distortion is negligible. The appearance or distortion could be observed on the monitor oscilloscope, and the amount of second and third harmonic generated could also be measured by the resolver. The pressure changes used were ± 5–10 mm Hg and the distortion was insignificant. This represents radius changes of less than ± 2–3 %.

The dynamic elastic modulus (E') is given by

$$E' = \frac{\Delta P}{\Delta R_o} \frac{2(1-\sigma^2)R_o R_i^2}{R_o^2 - R_i^2}$$

(see Bergel, 1961, preceding paper). E' may be resolved into two components, elastic and viscous. Following Hardung (1953) and McDonald & Taylor (1959) these are termed E_{dyn} and $\eta\omega$ respectively, and are defined as follows:

$$E_{dyn} = E' \cos \phi,$$
$$\eta\omega = E' \sin \phi,$$

where ϕ is the phase angle between pressure changes (ΔP leading) and radius changes (ΔR_o), $\eta\omega$ is the product of the coefficient of viscosity (η) and the angular velocity (ω), but only in a simple visco-elastic material is η constant.

Thus the amplitude of E', $|E'| = [(E_{dyn})^2 + (\eta\omega)^2]^{\frac{1}{2}}$, so that where $\eta\omega$ is small, say $< 10\%$ E_{dyn}, $E_{dyn} \simeq E'$.

Repeated determinations of these two quantities on rubber tubes suggest an over-all error of ± 5 %.

RESULTS

Some of the results have been briefly discussed previously (Bergel, 1960*a*). The behaviour of all the specimens was qualitatively similar, though there was wide quantitative variation between vessels of the same type. Table 1 shows the mean values of E_{dyn} for the four types of artery. It will be seen that all vessels showed an abrupt increase in stiffness between frequencies of 0 (i.e. static) and 2 c/s; with increasing frequency there was little further change. This increase appears to be greater for the more muscular vessels. This is well seen when the modulus ratio (E_{dyn}/E_{static}) is plotted against frequency (Fig. 2). This ratio correlates well with the muscularity of the vessel, though the variation is so great as to render the differences rarely significant (the vertical lines indicate the standard errors).

In all cases the viscous component ($\eta\omega$) was relatively small and again the relation to frequency was similar in all. At 2 c/s $\eta\omega$ was about 0·1 E_{dyn} and it increased by only about 1·5–2 times at 18 c/s. Since $\eta\omega = E' \sin \phi$ its magnitude depends on that of the complex modulus itself. This can be seen in Fig. 3 (top half) where it appears that the size of $\eta\omega$ also depends on the muscularity of the wall. However, the relation between E_{dyn} and $\eta\omega$ was remarkably similar for all specimens. Since $\phi = \tan^{-1} \eta\omega/E_{dyn}$ the variation of ϕ with frequency has been plotted in the lower half of Fig. 3. It can also be seen in this figure that the phase angle was very

TABLE 1. Mean values of E_{dyn} (dynes/cm^2 ± S.E. of means)

(Figures in brackets give the number of specimens.)

Vessel	0 c/s	2 c/s	5 c/s	18 c/s
Thoracic aorta	4·4 ± 0·40 (10)	4·7 ± 0·42 (10)	4·9 ± 0·45 (10)	5·3 ± 0·80 (4)
Abdominal aorta	9·2 ± 0·94 (7)	10·9 ± 0·88 (7)	11·0 ± 0·82 (7)	12·2 ± 0·46 (4)
Femoral artery	9·0 ± 1·15 (5)	12·0 ± 0·81 (5)	12·0 ± 0·82 (5)	10·6 ± 1·39 (5)
Carotid artery	6·9 ± 0·48 (6)	11·0 ± 1·00 (6)	11·3 ± 0·99 (6)	11·5 ± 1·03 (6)

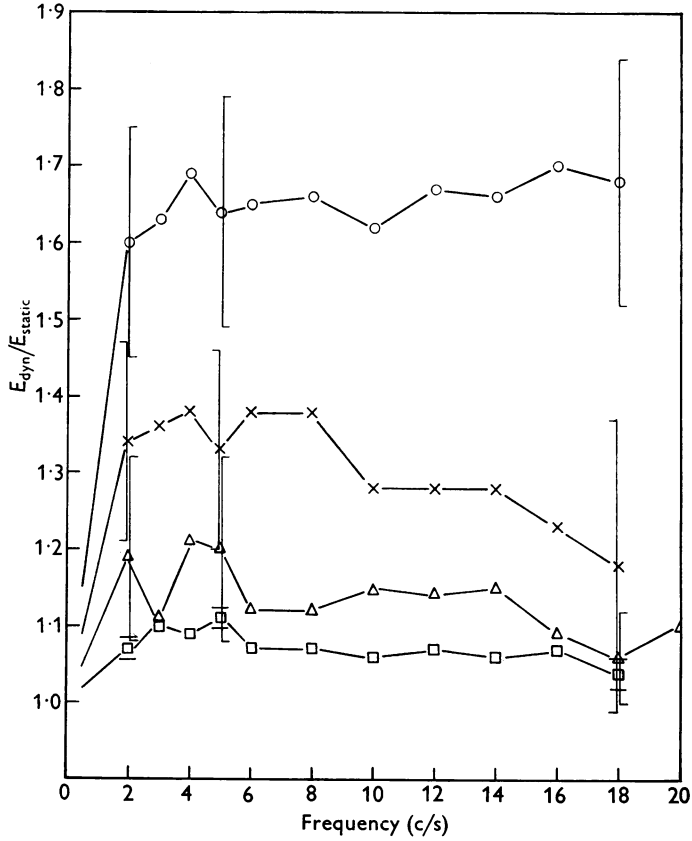

Fig. 2. Mean values for the relative modulus (E_{dyn}/E_{static}). Note the sharp increase between 0 and 2 c/s which correlates with the amount of smooth muscle in the vessel wall. The vertical lines show the standard errors. p values for the differences seen are as follows:

Difference between	2 c/s	5 c/s	18 c/s
Thoracic and abdominal	0·1	0·15	0·35
Abdominal and femoral	0·23	0·13	0·35
Femoral and carotid	0·1	0·1	0·05

□ thoracic aorta; △ abdominal aorta; × femoral artery; ○ carotid artery.

small (5–10°) and it could well have been overlooked entirely by merely inspecting the records of pressure and radius.

There was no difference in ϕ between different vessels at frequencies above 5 c/s, but at the lowest frequency it was rather smaller for the aortic specimens than for the other, more muscular, vessels. If the means

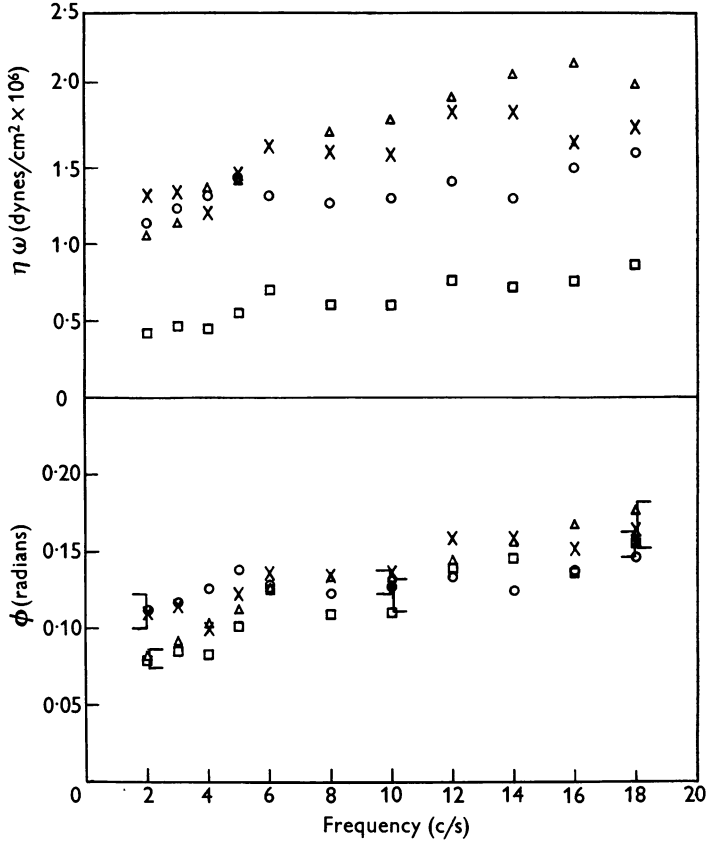

Fig. 3. Above: mean values of $\eta\omega$, the viscous component of the dynamic modulus. Below: mean values of ϕ, the phase angle between pressure and displacement. Note the great similarity between all vessels, except at the lowest frequencies, and the small change in ϕ between 5 and 18 c/s. The vertical lines show S.E. of means for the grouped means of the thoracic and abdominal aorta and the femoral and carotid arteries. □ thoracic aorta; △ abdominal aorta; × femoral artery; ○ carotid artery.

and standard errors for these two groups of artery are computed, a significant ($0{\cdot}02 > P > 0{\cdot}01$) difference appears between them at 2 c/s only. The standard errors for the two groups are shown in Fig. 3 (bottom half).

A few measurements were made at mean pressures of 50 and 150 mm Hg. The scatter was wide, but it appeared that the modulus ratio was independent of mean strain.

DISCUSSION

These results differ in some respects from those previously reported. Hardung (1953), studying aortic strips, found a modulus ratio of 1·25 at 2 c/s which did not alter up to 15 c/s. At low frequencies $\eta\omega$ was about 0·1 E_{dyn}, but thereafter it rose linearly with increasing rate of strain. Kapal (1954) with a similar technique and using human specimens, reported that the ratio increased steadily to 1·3 at 40 c/s (mean of 22). No measurements of phase differences were made but inspection of his records shows that they were no larger than those reported here. Kapal also found that the modulus ratio was not altered by the mean strain, but Lawton (1955) was unable to confirm this.

The rise in $\eta\omega$ seen by Hardung, and increase in E' found by Kapal, may be due to the use of strips rather than intact vessels. Ranke (1934) reported a very great increase in arterial viscosity when the specimens were immersed in hypertonic saline. This effect was reversible and was presumably due to removal of water from the tissues. The whole vessels filled with saline which were used in this work were always kept moist by seepage from tiny branches, but it is clearly considerably more difficult to maintain constant hydration of strips suspended in air.

A different approach is due to Lawton (1955, 1960) who has calculated the dynamic modulus from measurements of the natural frequency and rate of decay of oscillations in loaded longitudinal strips. These specimens were suspended in air at 25° C and 100% humidity. Lawton's results show that the static modulus and modulus ratio were similar (*ca.* 2×10^6 dynes/cm^2 and 2–2·5 respectively) in the aorta, iliac and carotid arteries of the dog, though the modulus ratio tended to be rather greater in the carotid. The rather low moduli might be expected from somewhat retracted strips (Bergel, 1961) but this cannot acccount for the similar ratios. Lawton (personal communication) has suggested that this might be due to the storage of his specimens in an ice-box for 24–36 hr before the experiment. It will be seen that the modulus ratio probably depends on the amount of muscle in the artery, a tissue which would be very sensitive to differences in experimental method.

Measurements made *in vivo* of the relationship between arterial pressure and diameter have been reported recently (Peterson *et al.* 1960). The dynamic modulus was found to be essentially constant over a range of about 2–10 c/s with values rather greater than those reported here. The viscous modulus was very much smaller, though it is not clear to what extent

it varied with frequency. There was a small phase difference between pressure and dilatation, with a delay usually less than 5 msec, though the authors were not very confident of the accuracy of determination of these very short times. This delay would represent a phase lag of 0·06–0·3 radians, very similar to that found here *in vitro*. Muscular activity has been shown both by Peterson *et al.* (1960) and by Hardung & Laszt (1960) to increase both the viscous and elastic components of the dynamic modulus and this may account, partly at least, for the rather high modulus measured *in vivo*. The excised specimens considered in this paper showed occasional spontaneous muscular contractions but it is likely that the over-all level of tone was very low. Nevertheless, it will be seen that the values reported here predict reasonable values for the pulse-wave velocity in the vessels concerned, while the results of Peterson *et al.* suggest velocities considerably higher than those generally accepted.

The behaviour seen in the arteries is not to be expected from any simple mathematical model embodying only viscous and elastic properties and not those due to inertia. Two lines of evidence suggest that this omission is reasonable. First, the computed resonant frequency for the thoracic aorta would not be lower than 300 c/s, very much higher than the frequencies actually employed. In the case of the other vessels the resonant frequency must be considerably higher. Secondly, inertial effects may be entirely discounted in the arterial stress-relation experiments of Zatzman Stacy, Randall & Eberstein (1954). A digital computer has been used to calculate the dynamic response of a blood vessel in which the pressure decayed in the manner found by these workers. Within the limits of the data fed to the computer the predicted behaviour fitted well with that actually observed.

Attempts were therefore made to fit the observed properties to those shown by a number of simple models composed solely of viscous and elastic elements in various combinations. These attempts were not successful. Possibly a much more complex model could be devised which would prove suitable, but the biological realities underlying this would not be readily apparent. This applies particularly to the concept of muscle viscosity and it is doubtful whether at the present time the construction of complex mathematical models of this sort serves any useful purpose.

The main conclusion to be drawn from Fig. 2 is that the modulus ratio ($E_{\mathrm{dyn}}/E_{\mathrm{static}}$) depends on the amount of muscle in the arterial wall (since $\eta\omega = ca.\ 0{\cdot}1\ E_{\mathrm{dyn}}$ there is little error in taking E_{dyn} as the dynamic modulus). Figure 3 further suggests that the viscous component in all these vessels is of a similar nature, and that it too is related to the smooth muscle.

In Figure 2 it can be seen that there is a relatively narrow frequency range in which the elastic modulus changes from the static to the dynamic value. This is known as the critical frequency, and for all arteries it is clearly below 2 c/s. Some experiments of Hardung (1953) at very low frequencies suggest it lies below 0·5 c/s. The critical frequency for resting molluscan smooth muscle has been determined (Pryor, 1952) and found to be below 0·02 c/s. This indicates that smooth muscle will show relatively long time constants for creep and stress relaxation and it has been reported by Abbot & Lowy (1957) that after a passive stretch tension decay in molluscan unstriated muscle was a double exponential with time constants of 5·8 and 68 sec. It may be concluded that the dynamic properties of arteries are dominated by the behaviour of smooth muscle but that the properties of muscle are too complex to be characterized by a simple spring-dashpot model.

If the change in modulus over the critical range is due to the inability of a smooth-muscle component to respond to rapid changes in tension it should be possible to estimate the contribution of the muscle to the modulus of the arterial wall. From data given by Reichel (1952) one may calculate values of 2·5 and $4{\cdot}5 \times 10^6$ dynes/cm^2 for the static and dynamic moduli respectively of resting molluscan muscle, i.e. a relative modulus of about 2. Assuming that this is the case for vascular smooth muscle, and that the femoral artery contains about 75% smooth muscle, and the carotid about 90% (unpublished observations) it appears that resting smooth muscle in the arterial wall has a static elastic modulus of $1\text{–}1{\cdot}5 \times 10^6$ dynes/cm^2. This calculation cannot be done for the aorta owing to the more complex structure of that vessel. The many assumptions made may well be in error, but this figure is in the range of values quoted in the literature for various types of unstriated muscle and a similar figure was suggested by static inflation experiments (Bergel, 1961).

The predicted pulse-wave velocity (p.w.v.)

The velocity of propagation of a pressure wave (m/sec) in a fluid-filled, thin-walled elastic tube is given by the Moens–Korteweg equation:

$$c = (Eh/2R\rho)^{\frac{1}{2}},$$

where E is the elastic modulus of the tube wall (dynes/cm^2),

ρ is the density of the fluid,

$h/2R$ is the relative wall thickness, the ratio of wall thickness (h) to the mean diameter ($2R$).

The relative wall thickness is somewhat greater in the smaller vessels (Bergel, 1961) but this increase is not enough, by itself, to account for the greater p.w.v. in these arteries.

This equation describes the behaviour of waves in a very thin-walled elastic tube filled with an incompressible non-viscous fluid, the radius being assumed to be very small compared with the wave-length. A full account of these factors is given by Lambossy (1950, 1951). The errors introduced by this last assumption are mostly very small, but the nature of the tube wall and the fluid viscosity are of more importance. The wall has been assumed to be an elastic boundary which increases in circumference alone when the pressure is raised, in effect taking the Poisson's ratio as zero. A further slight error is introduced by using the ratio of wall thickness to external radius rather than to the mean radius. An expression for the ratio of the true p.w.v. (c) to that obtained from the simple Moens–Korteweg equation (c'), in a tube of unaltering length can be derived (Bergel, 1960*b*). It is found that

$$\frac{c}{c'} = \left[\frac{2-\gamma,}{2-2\gamma(1-\sigma-2\sigma^2)+\gamma^2(1-\sigma-2\sigma^2)-2\sigma^2}\right]^{\frac{1}{2}},$$

where $\gamma = h/R_0$. It can be seen that when the wall is infinitely thin and $\sigma = 0$, $c = c'$. For arteries where $\gamma = 0{\cdot}1$–$0{\cdot}13$ and $\sigma = 0{\cdot}5$ the p.w.v. will be increased by about 12 %.

Wave propagation in a viscous fluid has been studied by Womersley (1957*a*) and the influence of wall viscosity on phase velocity and damping was discussed by Taylor (1959). Using their equations and employing the experimentally determined values for wall thickness and dynamic modulus, the p.w.v. and damping in these vessels have been computed. It is also assumed that the arteries are very stiffly tethered and loaded (Womersley, 1957*b*). This implies that they are unable to move longitudinally under the influence of the viscous drag from the blood flow. There is experimental evidence (Lawton & Greene, 1956) that the longitudinal movements of the wall are extremely small, though an untethered tube might be expected to show considerable movements (McDonald, 1960).

Figure 4 shows the computed values for the phase velocity and for the transmission (fraction of wave amplitude transmitted) over a distance of 10 cm. In the presence of reflexions the phase velocity can be considerably altered but this effect will be less marked for high-frequency, short wave-length oscillations (McDonald & Taylor, 1959). The foot-to-foot p.w.v. is that of the high-frequency components responsible for the sharp inflexion at the foot of the pulse wave and should therefore be compared with the phase velocity predicted for the higher frequencies.

Measured pulse-wave velocities have been fully listed previously (Bergel, 1960*b*). The following figures give the range of the reported values: thoracic aorta, 4·5–7 m/sec; abdominal aorta, 5·5–8·5 m/sec; femoral artery, 8–12 m/sec. Though no measurements of carotid p.w.v. have been

reported, the *in vitro* experiments of Bramwell, Downing & Hill (1923) suggest that it is similar to that in the femoral.

The calculated figures are in good agreement with those quoted. The predicted velocity in the abdominal aorta is high and that for the femoral

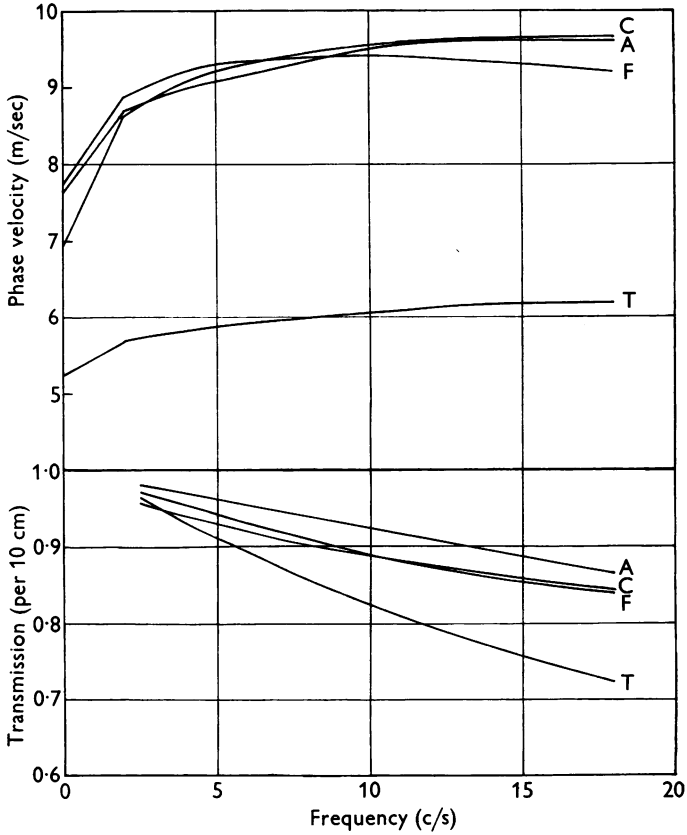

Fig. 4. Above: variation of phase velocity with frequency. Below: variation of wave transmission (amplitude of downstream oscillations/amplitude of upstream oscillations) over a distance of 10 cm. All these curves are computed from the experimental results. The letter at the end of each curve indicates the type of artery. The range of reported pulse-wave velocities is as follows: thoracic aorta 4·5–7 m/sec, abdominal aorta 5·5–8·5 m/sec, femoral artery 8–12 m/sec.

and carotid arteries lies at the bottom of the range, but it has been shown that muscular activity will increase the velocity by increasing both E_{dyn} and $\eta\omega$ (Hardung & Laszt, 1960; Peterson *et al.* 1960). There is little direct evidence on the rate of damping of the pulse wave, which cannot be measured from changes in amplitude if reflexions are present. The transmission is very low in the thoracic aorta, for in this vessel 10 cm is a

considerable fraction of the high-frequency wave-length. Certainly the sharp dicrotic notch seen in recordings from this vessel is damped out very rapidly. Landowne (1957) has studied the transmission of superimposed pressure spikes in human brachial arteries; in this case reflexions have no effect. Taking reasonable values for the distance separating his two recording points and for the dimensions of the artery the predicted reduction in amplitude is about 60 %, and this is in fact the reduction seen in the reproduced pressure recordings.

SUMMARY

1. The visco-elastic properties of dog's arteries from four anatomical sites have been studied by measuring the dynamic pressure–radius relationships at frequencies up to 20 c/s.

2. There is an abrupt increase in the dynamic elastic modulus between 0 and 2 c/s, and the size of this increase correlates with the muscularity of the vessel wall. With increasing frequency the dynamic modulus remains essentially constant.

3. The phase angle between pressure and dilatation is very small (less than 10°) and it alters with frequency in a manner different from that predicted by the use of simple mechanical models containing elastic and viscous elements.

4. The pulse-wave velocity and the amount of damping have been calculated for these arteries. The values reported in the literature compare satisfactorily with those computed from the experimental results.

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